Posterior myocardial infarction: the dark side of the moon

Case report and review of electrocardiographic diagnosis

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The clinical presentation of posterior myocardial infarction is not always easy, not even for the cardiologist. In this article a 70-year-old woman who presented with chest pain is described. The electrocardiogram at presentation showed marked ST-segment depression in leads V_1 to V_5 and slight ST-segment depression in leads I and aVL. There was ST-segment elevation in the posterior leads V_7 to V_9. Elevation of specific cardiac enzymes confirmed the diagnosis of myocardial infarction. True posterior myocardial infarction is difficult to recognise because the leads of the standard 12-lead electrocardiogram are not a direct representation of the area involved. Only with indirect changes in the precordial leads as such the diagnosis can be suspected. This review will highlight the electrocardiographic fine-tuned diagnosis of posterior myocardial infarction by using the posterior leads V_7 to V_9 leading to easier and faster recognition with consequences for treatment and improved prognosis. (*Neth Heart J* 2007;15:16-21.)

Keywords: myocardial infarction (posterior), electrocardiogram, diagnosis

True posterior myocardial infarction (PMI), the 'dead angle infarction' of the electrocardiogram (ECG), is often misjudged and this may be the reason for undertreatment. It is suggested to be one of the most commonly missed types of acute myocardial infarction (MI) electrocardiographic patterns.[1] The clinical presentation of PMI is not different from other myocardial infarctions, but the absence of 'traditional' electrocardiographic infarct signs such as ST-segment elevation can lead to errors or delay in the diagnosis. Correct interpretation and use of the ECG using the additional leads V_7 to V_9 can establish the electrocardiographic diagnosis of PMI.

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Case report

A 70-year-old woman with a history of thyroid disease, polymyalgia rheumatica and hypercholesterolaemia was admitted to the emergency department. She had complaints of acute chest pain with radiation to the left shoulder, dyspnoea and nausea. On physical examination an adipose woman (BMI 36.2 kg/m^2) was seen. Blood pressure was 144/70 mmHg and pulse 77 beats/min. On auscultation normal heart sounds and a midsystolic murmur grade II/VI were heard. Complete physical examination revealed no further abnormalities.

The ECG showed sinus rhythm with a tall R wave in lead V_2, minimal ST-segment elevation in lead III and ST-segment depression in leads V_2 to V_5, I and aVL (figure 1). There were ST-segment elevations in the posterior leads V_7 to V_9 (figure 2). Chest X-ray revealed no abnormalities. The patient was at that point in time being treated with a thrombolytic agent because of the clinical and electrocardiographic findings. She was also given aspirin, metoprolol and enoxaparin. She was already taking atorvastatin, levothyroxin and prednisone. Thrombolysis was followed by rapid normalisation of the ST segments (figure 3) and cessation of chest pain. Laboratory results confirmed the diagnosis of MI: troponin-T 0.59 µg/l (N<0.04 µg/l), maximum creatine kinase (CK) 1748 U/l (N=0-200 U/l), and maximum aspartate aminotransferase (ASAT) 199 U/l (N=0-40 U/l). On discharge the patient was on aspirin 80 mg, metoprolol 200 mg, atorvastatin 40 mg, prednisone and levothyroxin.

Two months later, the patient was readmitted to the hospital with chest pain. The hospital diagnosis was a

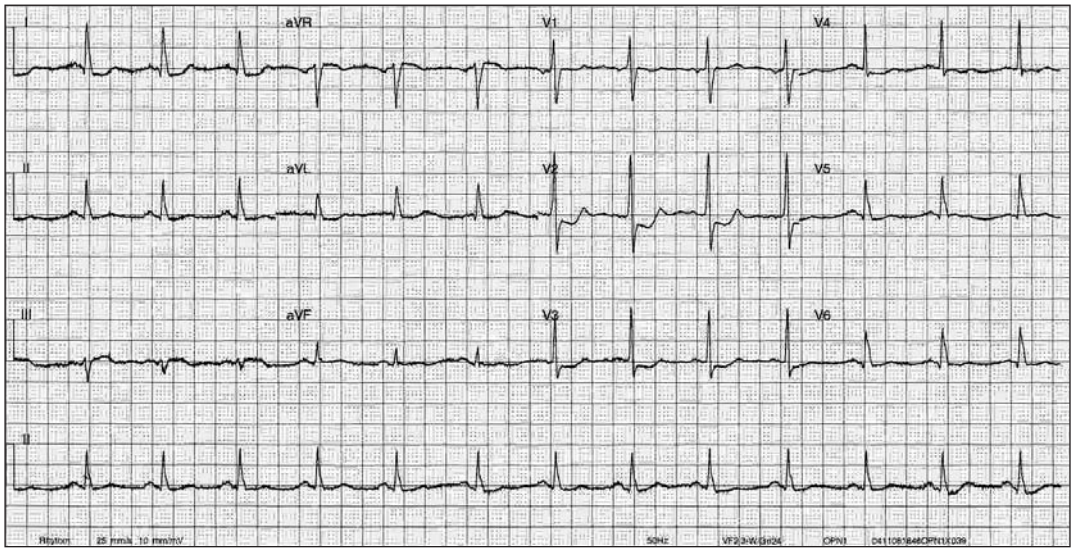


Figure 1. 12-lead ECG on admission.

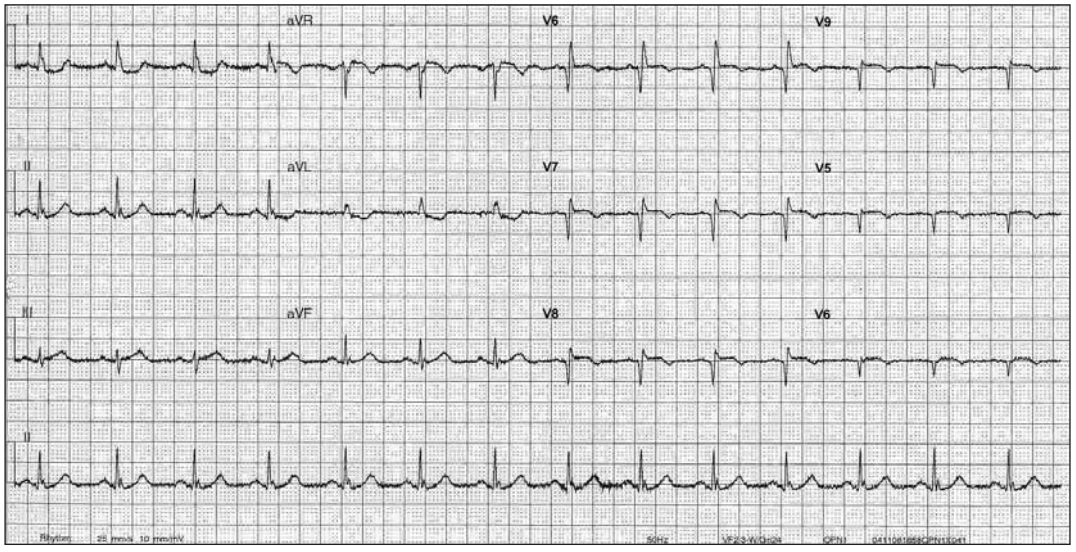


Figure 2. Posterior leads V_7 to V_9 on admission.

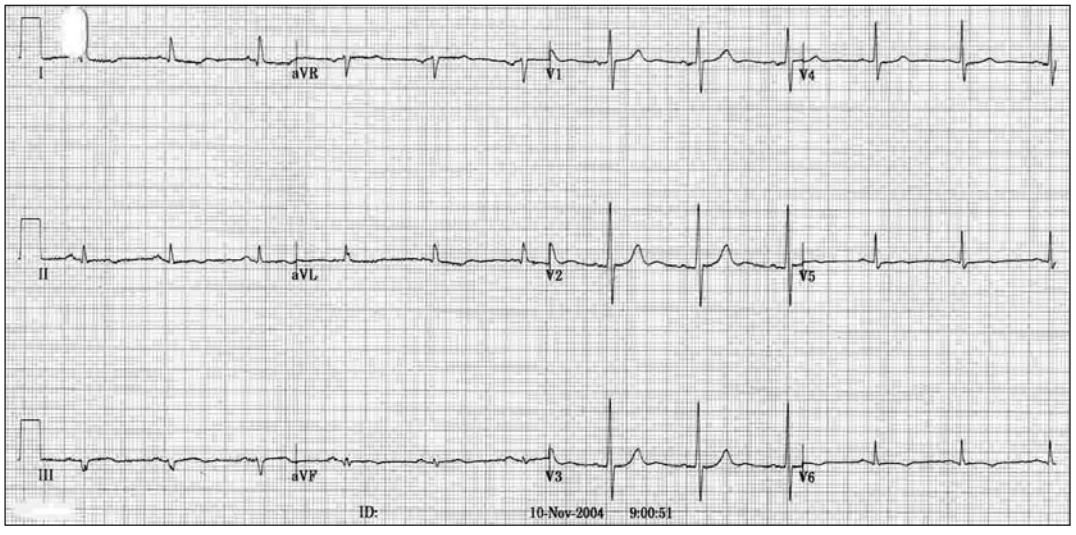
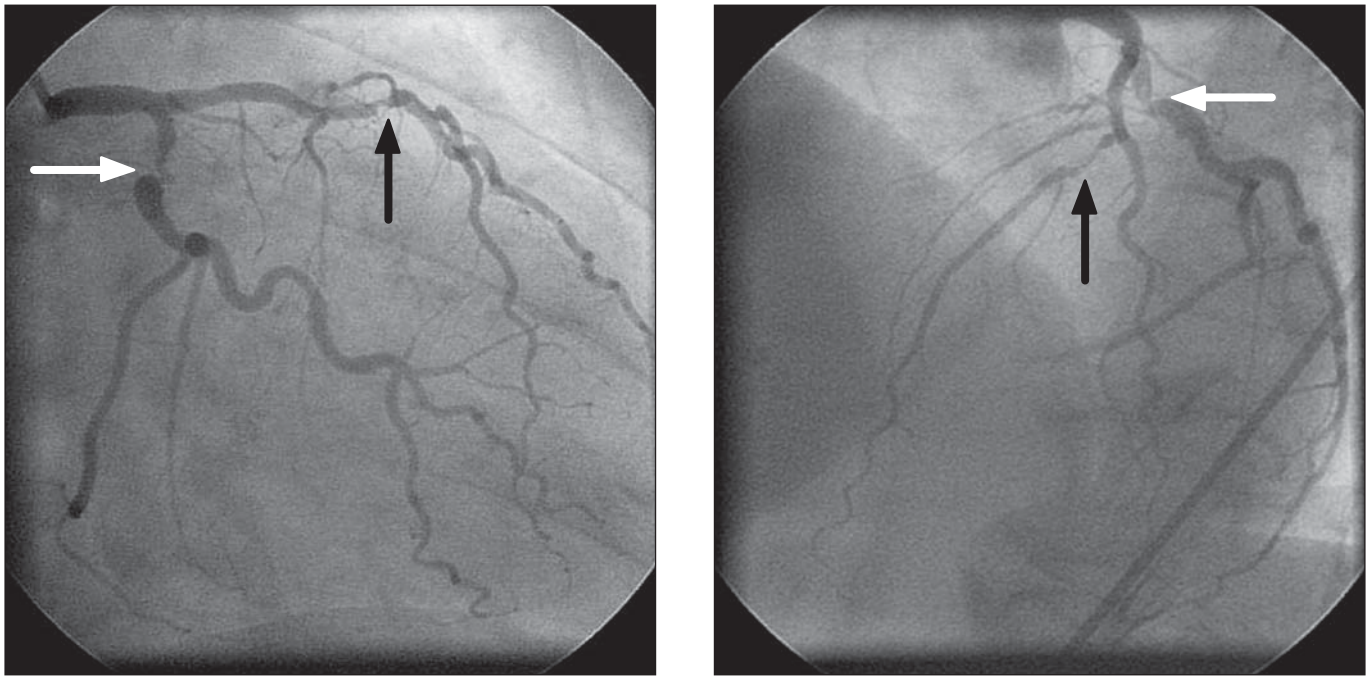


Figure 3. ECG two days after thrombolysis.



Figures 4 and 5. Coronary angiography: right anterior oblique view (left picture) and left anterior view (right picture), left coronary artery, showing a 90% stenosis of the circumflex artery (white arrow) and a 70% stenosis in the left anterior descending (black arrow) artery just distal to a large diagonal branch.

non-ST-elevation myocardial infarction (NSTEMI), with troponin-T 0.74 µg/l, maximum CK 1012 U/l and maximum ASAT 134 U/l. Exercise testing before discharge showed significant ST-segment depression suggestive of ischaemia. At coronary angiography (figures 4 and 5) three-vessel coronary artery disease was diagnosed, with an ostial stenosis in the right coronary artery (RCA), a 90% stenosis in the large circumflex artery (RCX) and a 70% stenosis in the left anterior descending artery (LAD). Because of obesity, percutaneous coronary intervention of the RCX and LAD was performed instead of coronary bypass grafting. The mid LAD and proximal RCX were stented. The procedure was uneventful.

Discussion

The term PMI is used for necrosis of the dorsal, infra-atrial part of the left ventricle located beneath the atrioventricular sulcus. The majority of patients with the typical electrocardiographic abnormalities for PMI have a stenosis or occlusion of the RCX.[1-3] Some patients have a stenosis or occlusion of the RCA. In 10% of the population the RCX is the dominant vessel. It is the least commonly infarcted coronary artery.[3] PMI has been reported to represent 15 to 21% of acute MI, often accompanied by inferior and/or lateral MI.[4-6] A strict and true PMI is thought to be very rare; however, a more recent study showed an incidence of 3.3% using posterior leads.[7] Risk factors, clinical presentation and differential diagnosis are no different than with other myocardial infarctions.

Rapid recognition of acute PMI is important for several reasons. Patients with inferior or lateral MI who also have PMI have a larger-sized infarct with an increased risk of complications, such as left ventricular dysfunction and death.[7,8] In a group of 33 consecutive patients with ST-segment elevation only in leads V_7 to V_9, 22% of patients had moderate to severe mitral regurgitation.[9] Patients with inferoposterior MI may benefit more from reperfusion therapy than patients with myocardial infarction of a 'single wall'. Patients with electrocardiographic isolated PMI often do not receive the appropriate reperfusion treatment if the clinical diagnosis of MI is not suspected, probably due to lack of the classical ST-segment elevation.[4]

Electrocardiographic findings

Electrocardiographic findings of PMI are shown in table 1. The electrocardiographic diagnosis is difficult because no specific leads of the standard ECG directly represent this area.[10] There is loss of the electrical forces in a dorsal direction, so that a typical infarction pattern only appears in the electrodes placed dorsally between the spine and left scapula on the ECG. On the standard ECG of true PMI the leads V_1 and V_2 are a mirror image of the V_1 and V_2 leads of the anterior MI.[1,10]

QRS complex

During PMI the QRS complex on the vector cardiogram points ventrally due to losses of the electrical forces normally aimed dorsally, resulting in a prolonged R wave. An increase in the R/S ratio >1.0 occurs in

Table 1. Electrocardiographic criteria of PMI.

Standard 12-lead ECG
ST-segment depression (horizontal >> downsloping/upsloping)*
Prominent R wave*
R/S wave ratio >1.0 in lead V_2
Prominent, upright T wave*
Combination of horizontal ST-segment depression with upright T wave*
Co-existing acute inferior and/or lateral MI
- Additional lead ECG (posterior leads V_7 to V_9)
≥ 1 mm ST-segment elevation

* Limited to leads V_1 to V_3. Adjusted from Brady et al.[11]

leads V_1 and V_2 as PMI evolves.[1,4,10] The increase of the R wave in PMI is the opposite to the Q wave associated with traditional ST-segment elevated myocardial infarction. In some patients the increase of the R wave in lead V_1 results from a conduction failure in the His-Purkinje system which is located in the left fascicle of the septum. This suggestion is supported by the fact that about 30% of patients with a PMI develop second- or third-degree atrioventricular block due to ischaemia.[12] A tall R wave in leads V_1 and V_2 on the ECG is not only seen in right ventricular hypertrophy, hypertrophic cardiomyopathy, right bundle branch block, Wolff-Parkinson-White syndrome, Duchenne muscle dystrophy, right ventricle infarction and posterolateral left wall infarction but also in healthy children and healthy people with prominent forward QRS vectors (table 2). However, with the use of clinical symptoms, additional investigation for these disorders can distinguish PMI.[1,12,13]

ST segment

The ST segment points in the direction of the infarcted area and ST-segment depression occurs in the precordial leads in the acute phase. The latter is also seen in precordial ischaemia or as reciprocal changes in MI. The T wave points away from the infarcted area. As a result a forward movement of the T wave can frequently be seen in patients with PMI. The combination of right precordial horizontal ST-segment depression with tall, upright T waves indicates an early electrocardiographic sign of acute ischaemia of the posterior wall during a progressive PMI.[1,2] The movement of the T wave on the ECG can be ascertained by the T_2 to T_6 index which is the difference between the voltages of the T waves in leads V_2 and V_6. If the value of the T_2 to T_6 index equals or extends the value of 0.38 mV, then the probability of PMI is more than likely.[1,12,13]

Use of dorsal leads V_7 to V_9

Mortality reduction is highest when reperfusion of the infarcted vessel is achieved within six hours of pain onset, with the best results during the first 'golden' hour.[15-17] In search of faster and more reliable methods in identifying PMI, the extra posterior leads V_7 to V_9 significantly increase the detection of posterior injury patterns compared with the standard 12-lead ECG.[10] Lead V_7 should be placed at the level of lead V_6 at the posterior axillary line, lead V_8 on the left side of the back at the tip of the scapula and lead V_9 is placed halfway between lead V_9 and the left paraspinal muscles. ST-segment elevation of >1 mm in the posterior leads is suggestive of PMI.[18] Using all 15 leads significantly improves the further detection of circumflex coronary-related injury pattern over the standard 12-lead ECG.[2,9,19] Sensitivity increased from 32 to 57% with a specificity of 98% for the circumflex artery.[19] Five percent of diagnosed non-Q-wave MIs are retrospectively PMIs in which reperfusion therapy would

Table 2. Differential diagnosis of tall R waves in right precordial leads.

Diagnosis	**Confirmatory clues**
True posterior infarct	ST↓, T↑ in V_1-V_2; Q waves and ST↑ V_7 to V_9
Right ventricular hypertrophy	RAD, RAE; secondary ST-Ts; V_7 to V_9 normal
Ventricular septal hypertrophy	Associated Q waves; LVH; V_7 to V_9 normal or deep narrow Q waves
Right bundle branch block	Wide QRS; broad S in V_1, V_6; R peaks late in V_1; V_7 to V_9 normal or broad S waves
Wolff-Parkinson-White syndrome	Short PR; delta wave; V_7 to V_9 normal or delta wave
Normal variant	No other abnormalities

LVH=left ventricular hypertrophy RAD=right-axis deviation, RAE=right atrial enlargement. Adjusted from Casas et al.[14]

have been justified if posterior leads had been recorded.[4] The sensitivity and positive predictive value for the diagnosis of MI with the additional leads V_7 to V_9 and right ventricular leads V_{4R} to V_{6R} increases from 57.7 to 66.1% and from 88.4 to 96.8%, respectively, but the specificity for MI decreases from 91 to 84%. The overall accuracy of the additional-lead ECG is only modestly improved.[6] A study investigating the use of 15-lead ECG in comparison with 12-lead ECG in every emergency chest pain patient showed no alterations in diagnosis and management.[5] It is advised to only use the 15-lead ECG in a subgroup of patients more likely to have PMI or right ventricular infarction. There is an increase in reported incidence of PMI using the 15-lead ECG compared with the 12-lead ECG, suggesting more undiagnosed PMI using the 12-lead ECG.[20] Kulkarni et al. prospectively showed the electrocardiographic changes during RCA and RCX balloon inflation, mimicking an occlusion in the related artery.[21] They found that ST-segment elevation restricted to V_7 to V_9 was only seen during RCX inflation. Inferior elevation alone was not seen with RCX inflation. Patients with ST-segment elevation in the inferior and posterior leads are more likely to have RCX occlusion rather than RCA (85%). Schmitt et al. found the sensitivity of the ECG diagnosis of AMI was increased by 11% (from 50 to 61%) for RCX lesions by extended precordial leads, after comparing ECG diagnosis with angiographic findings.[22] They found a trend toward an extended infarct size in those patients with concomitant ST-segment elevation in the additional ECG leads.

There are no studies showing improved outcome after thrombolysis in PMI. Thrombolysis has potential hazardous complications so there should be awareness for pseudo infarct patterns. As with any MI it is important to start reperfusion treatment as soon as possible when there is a strong suspicion for PMI based on clinical signs, symptoms and electrocardiographic findings.

In the early stage of acute MI the ECG may be normal or near normal. Less than 50% of patients with acute MI have clear changes suggestive of the diagnosis on their first ECG. About 10% of laboratory-proven MIs are NSTEMI.[23] Thrombolysis is justified in patients with >1 mm ST elevation in two contiguous leads in the limb leads or >2 mm ST elevation in two contiguous chest leads or in patients with new left bundle branch block.[16,23-26] Fibrinolytic therapy may be appropriate when there is marked ST-segment depression confined to leads V_1 to V_4 accompanied by tall R waves in the right precordial leads and upright T waves indicative of true posterior injury. Confirmatory data from posterior leads may be especially helpful.[26] It is suggested that because of the greater distance between the infarcted area and the leads in PMI, an elevation of 0.5 mm is sufficient to justify the diagnosis of PMI followed by decisions in reperfusion treatment. Adjusted criteria provided an improved sensitivity from 49% in the 12-lead to 94% in 15-lead ECG.[27] An increase of the use of thrombolysis was seen with the increasing number of electrocardiographic leads demonstrating ST-segment elevation.[6] Posterior chest leads should be routinely recorded in patients with suspected MI and nondiagnostic routine ECG to establish the appropriate reperfusion treatment (including thrombolysis) of some of the patients now classified as having unstable or non-Q-wave MI.[2] Some authors claim that in specific cases ST-segment depression in the precordial leads is sufficient for thrombolysis.[28] By using the posterior leads V_7 to V_9 this uncertainty will not be necessary in most cases.[4] Primary angioplasty has the advantage of establishing the diagnosis and therapy immediately. It should be emphasised that patients presenting with MI with co-existing PMI are at greater risk of complications, and acute therapy including thrombolysis and angioplasty should not be delayed.

Conclusion

PMI is responsible for subtle changes on the ECG. For the diagnosis of PMI it is important to recognise the clinical signs at presentation, combined with subtle manifestations on the ECG in order to start reperfusion therapy early. Cardiologists should be able to interpret electrocardiographic signs such as ST-segment depression with upright T waves and prominent tall R waves in leads V_1 to V_3 in order to make sure that a posterior lead ECG is recorded in these cases. Using posterior leads in patients presenting with symptoms suspicious for MI will reveal more patients with PMI who will benefit from early reperfusion treatment. Retrospective analysis of available data concerning non-ST-segment ASC might give new insights in the indication of PMI. ■

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